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Health Effects Assessment for Naphthalene

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HEALTH EFFECTS ASSESSMENT
FOR NAPHTHALENE

U.S. Environmental Protection Agency
Office of Research and Development
Office of Health and Environmental Assessment
Environmental Criteria and Assessment Office
Cincinnati, OH 45268

U.S. Environmental Protection Agency
Office of Emergency and Remedial Response
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16. ABSTRACT This document represents a brief, quantitatively oriented scientific summary of health effects data. It was developed by the Environmental Criteria and Assessment Office to assist the Office of Emergency and Remedial Response in establishing chemical-specific health-related goals of remedial actions. If applicable, chemical-specific subchronic and chronic toxicity interim acceptable intakes are determined for systemic toxicants, or q_1^* values are determined for carcinogens for both oral and inhalation routes. Sufficient data are not available at this time on naphthalene to determine either acceptable intakes or q_1^*s.		
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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with naphthalene. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to September, 1984. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) source have been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA-440/5-80-059. NTIS PB 81-117707.

U.S. EPA. 1982. Revision and Update of Hazard Profile on Naphthalene. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the available data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, the AIS or acceptable intake subchronic, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for AIS estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure.

The AIC, acceptable intake chronic, is similar in concept to the ADI (acceptable daily intake). It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980b) for a discussion of this concept]. The AIC is route specific and estimates acceptable exposure for a given route with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities; the methodology for their development is explained in U.S. EPA (1983b).

For compounds for which there is sufficient evidence of carcinogenicity, AIS and AIC values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980b). Since cancer is a process that is not characterized by a threshold, any exposure contributes an increment of risk. Consequently, derivation of AIS and AIC values would be inappropriate. For carcinogens, q1*s have been computed based on oral and inhalation data if available.

ABSTRACT

In order to place the risk assessment in proper context, the reader is referred to the preface of this document. The preface outlines limitations applicable to all documents of this series, as well as the appropriate interpretation and use of the quantitative estimates.

The absence of adequate toxicological data on naphthalene precludes estimation of acceptable intakes for any route.

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LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
AIC	Acceptable intake chronic
AIS	Acceptable intake subchronic
CAS	Chemical Abstract Service
CS	Composite score
ppm	Parts per million
RQ	Reportable quantity
STEL	Short-term exposure limit
TLV	Threshold limit value
TWA	Time-weighted average

1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of naphthalene (CAS No. 91-20-3) are as follows:

Chemical class:	polycyclic aromatic hydrocarbon
Molecular weight:	128.19 (Callahan et al., 1979)
Vapor pressure at 25°C:	0.082 mm Hg (MacKay et al., 1982)
Water solubility at 25°C:	31.7 mg/l (MacKay et al., 1980)
Log octanol/water partition coefficient:	3.37 (MacKay et al., 1980)
Bioconcentration factor:	146 (estimated from the equation of Veith et al., 1979)
Half-life in air:	0.7 (Atkinson et al., 1984)
Half-life in water:	~1 day (Callahan et al., 1979) 2.3 days (Zoeteman et al., 1980)
Half-life in soil:	<1 day (estimated)

The half-life for naphthalene in aquatic media caused by biodegradation can be estimated to be ~14 hours from the estimated biotransformation rate constant (1×10^{-7} ml cell⁻¹ hr⁻¹) reported by Mabey et al. (1981) and the concentration of microorganisms at 5×10^5 cell ml⁻¹ (Burns et al., 1982).

Pertinent data regarding the leachability of this compound in soils could not be located in the available literature. Considering the soil sorption coefficient (Kenaga and Goring, 1980) and the water solubility, this compound is expected to have higher mobility in soils compared with most other polycyclic aromatic hydrocarbons. The biodegradability of this compound in soils, however, is such that significant leaching into groundwater is not likely.

2. ABSORPTION

2.1. ORAL

Information regarding the absorption of naphthalene in humans and animals is limited. When ingested as a solid, naphthalene is sufficiently absorbed to cause toxicity in man (Chusid and Fried, 1955; Zuelzer and Apt, 1949; Nash, 1903; Gross et al., 1958; Haggerty, 1956). Furthermore, naphthalene seems to be more toxic when dissolved in oil before ingestion (Talakin, 1966). Sanborn and Malins (1977) suggested that naphthalene may be absorbed to a greater extent when ingested in water than in food.

2.2. INHALATION

Data regarding the absorption of inhaled naphthalene are limited; however, Valaes et al. (1963) reported toxicity and death in newborn infants exposed to naphthalene vapors from clothes or blankets that had been stored in or near the infants' rooms.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. Data regarding subchronic oral exposure to naphthalene are limited. Several investigators (Anziulewicz et al., 1959; Zinkham and Childs, 1958) reported the occurrence of hemolytic anemia in infants born to mothers who had "sniffed" and ingested unspecified quantities of naphthalene (in the form of mothballs) during pregnancy. The mothers themselves were also anemic, but to a lesser extent than were the infants.

Several animal studies focused upon ocular effects of naphthalene, but failed to mention whether any other signs of toxicity were measured or observed. Fitzhugh and Buschke (1949) observed slight cataracts in weanling rats exposed to 2% naphthalene in the diet for 60 days. Assuming a rat consumes food equivalent to 5% of its body weight/day, this dietary level is equivalent to a dose of 1 g/kg bw/day.

In two separate studies, rabbits exposed to 1 g naphthalene/kg bw/day by gavage (either in light paraffin or an unspecified vehicle) for ~46 days developed cataracts (Ghetti and Mariani, 1956; Van Heyningen and Pirie, 1976) and degeneration of the retina (Van Heyningen and Pirie, 1976) within the first few days of treatment.

3.1.2. Inhalation. Subchronic data regarding the inhalation of naphthalene are limited to two studies of occupational exposure. Van der Hoeve (1906) reported that one man who worked with powdered naphthalene developed cataracts and retinal hemorrhage, while another man exposed similarly had chorioretinitis. Ghetti and Mariani (1956) reported that 8/21 workers exposed to unspecified "high" concentrations of naphthalene in a dye-manufacturing process developed cataracts. Since these men were <50 years of age, it is unlikely that they would have developed cataracts spontaneously.

3.2. CHRONIC

3.2.1. Oral. Pertinent data regarding the chronic toxicity of orally administered naphthalene could not be located in the available literature.

3.2.2. Inhalation. Pertinent data regarding chronic toxicity of inhaled naphthalene could not be located in the available literature.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

3.3.1. Oral. Pertinent data regarding the teratogenicity of ingested naphthalene could not be located in the available literature; however, naphthalene or its metabolites are known to cross the placenta in sufficient quantities to cause fetotoxic effects (Anziulewicz et al., 1959; Van der Hoeve, 1913). These effects included hemolytic anemia in humans and cataracts and retinal damage in rabbits. Doses either were not estimated or were not reported in the secondary sources.

3.3.2. Inhalation. Pertinent data regarding the teratogenicity of inhaled naphthalene could not be located in the available literature.

3.4. TOXICANT INTERACTIONS

A woman exposed for 3 weeks to a combination of naphthalene and paradichlorobenzene (while mothproofing clothing) developed aplastic anemia 1 month after exposure (Harden and Baetjer, 1978). No other cases of aplastic anemia associated with exposure to either naphthalene or paradichlorobenzene alone have been reported in the literature.

4. CARCINOGENICITY

4.1. HUMAN DATA

4.1.1. Oral. Pertinent data regarding the carcinogenicity of ingested naphthalene could not be located in the available literature.

4.1.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled naphthalene could not be located in the available literature; however, Wolf (1976) reported that 6/15 workers exposed to vapors of both naphthalene and coal tar developed laryngeal carcinomas (4) or neoplasms of the pylorus and cecum (2). There was no control group. Schmeltz et al. (1978), however, showed that the di-, tri- and tetramethyl naphthalene contaminants of coal tar were carcinogenic when applied to mouse skin but that naphthalene alone was not.

4.2. BIOASSAYS

4.2.1. Oral. In a study designed to investigate the carcinogenic effects of anthracene delivered by a naphthalene vehicle, the carcinogenicity of naphthalene alone was tested (Druckrey and Schmahl, 1955). A group of 28 BD I and BD III strain rats were exposed orally to 10 g naphthalene/rat for an unspecified amount of time and monitored for >1000 days. A second group of 10 rats were exposed subcutaneously to 0.82 g naphthalene/rat for a similar period of time. No tumors were observed in either group. No other studies pertaining to the carcinogenicity of orally administered naphthalene were located in the available literature.

4.2.2. Inhalation. Pertinent data regarding the carcinogenicity of inhaled naphthalene could not be located in the available literature.

4.3. OTHER RELEVANT DATA

The genotoxic activity of naphthalene has been tested in two in vitro systems. Cell transformation was not seen in rodent embryo cells pretreated with leukemia virus and exposed to concentrations of naphthalene up to 100 mg/l of culture medium (Freeman et al., 1973; Rhim et al., 1974). Similarly, cell transformations were not seen in a murine mammary gland organ culture system at naphthalene concentrations up to 1000 mg/l of culture medium (Tonelli et al., 1979).

Naphthalene was not found to be mutagenic in a number of bacterial/microsomal assay systems (McCann et al., 1975; Kraemer et al., 1974).

4.4. WEIGHT OF EVIDENCE

IARC has not evaluated the risk to humans associated with oral or inhalation exposure to naphthalene. Applying the criteria for evaluating the overall weight of evidence for carcinogenicity proposed by the Carcinogen Assessment Group of the U.S. EPA (Federal Register, 1984), the evidence for carcinogenicity of naphthalene in humans and animals is inadequate and the chemical is most appropriately designated a Group D-Not Classified chemical.

5. REGULATORY STANDARDS AND CRITERIA

The ACGIH (1983) has recommended a TLV-TWA of 50 mg/m³ (10 ppm) and an STEL of 75 mg/m³ for occupational exposure to naphthalene. Since eye irritation was seen at 15 ppm, this criterion was chosen "to prevent ocular effects but possibly not blood changes in hypersusceptibles." The OSHA standard for exposure to naphthalene in the workplace is a TWA of 50 mg/m³ (Code of Federal Regulations, 1981).

6. RISK ASSESSMENT

6.1. ACCEPTABLE INTAKE SUBCHRONIC (AIS)

6.1.1. Oral. The only studies that defined exposure levels (Ghetli and Mariani, 1956; Van Heyningen and Pirie, 1976) failed to mention whether end-points other than ocular effects were monitored. Since other effects such as hemolytic anemia have been associated with exposure to naphthalene, it would not be prudent to calculate an AIS on the basis of these studies.

6.1.2. Inhalation. The lack of pertinent, quantitative data precludes assessment of risk caused by inhalation of naphthalene.

6.2. ACCEPTABLE INTAKE CHRONIC (AIC)

6.2.1. Oral. Data regarding chronic oral exposure to naphthalene were not located in the available literature. For reasons given in Section 6.1.1., an AIC cannot be derived from the subchronic data. Data from which a CS for oral exposure to naphthalene can be calculated have not been located in the available literature (U.S. EPA, 1983a).

6.2.2. Inhalation. Data regarding chronic exposure to naphthalene by inhalation could not be located in the available literature. Furthermore, it would be imprudent to use the TLV established by the ACGIH (1983) to calculate an AIC, since this value was established essentially on the basis of eye irritation and with the hypothesis that chronic exposure might cause more severe effects. Data from which a CS for inhalation exposure to naphthalene can be calculated have not been located in the available literature (U.S. EPA, 1983a).

6.3. CARCINOGENIC POTENCY (q_1^*)

6.3.1. Oral. Naphthalene was not carcinogenic in strain BD I and BD III rats (Druckrey and Schmahl, 1955). Furthermore, mutagenicity and in vitro bioassays that tested only naphthalene yielded negative results. It is therefore not possible to derive a q_1^* for oral exposure to naphthalene.

6.3.2. Inhalation. The lack of pertinent data regarding the carcinogenicity of inhaled naphthalene precludes assessment of carcinogenic potency.

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APPENDIX

Summary Table for Naphthalene

Species	Experimental Dose/Exposure	Effect	Acceptable Intake (AIS or AIC)	Reference
Inhalation				
AIS			ND	
AIC			ND	
Carcinogenic potency			ND	
Oral				
AIS			ND	
AIC			ND	
Carcinogenic potency			ND	

ND = Not derived